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Nerve growth factor delays retinal degeneration in C3H mice.

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Abstract

BACKGROUND: The aim of the present study was to investigate the biological role of nerve growth factor (NGF) on retinal degeneration in the C3H mouse strain. This strain is characterized by a single gene mutation (rd) which leads to photoreceptor degeneration resembling human retinitis pigmentosa. METHODS: Neural retinas from 1- to 25 day-old C3H mice were dissected from outer ocular tissues, dissociated in cell suspension, stained with a vital dye and counted in a hemocytometer. For in vivo study, NGF was injected into the intraocular or retro-ocular area, and at the end of the treatment the mice were killed. The eyes were enucleated, fixed and cut by cryostat into 14-microns serial sections. The serial sections were stained with hematoxylin-eosin and the outer nuclear layer (ONL) was measured using a computerized image analysis system. RESULTS: An intraocular injection of NGF, or repeated retro-ocular injections, induced a significant increase in ONL thickness compared to controls. CONCLUSION: Our data show that NGF inhibits retinal degeneration in C3H mice. The mechanism(s) underlying the protective action of NGF against retinal cell death remains to be established.

PMID: 8871157 [PubMed - indexed for MEDLINE]

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Nerve growth factor promotes functional recovery of retinal ganglion cells after ischemia.

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Abstract

PURPOSE: To investigate the effect of a transient complete ischemia on the function of cat retina and to determi whether nerve growth factor (NGF), which was previously shown to enhance retinal ganglion cell (RGC) survival optic nerve section in the adult rat, can promote recovery of retinal neurons after the ischemic insult. METHODS: Function of distal and proximal retina was assessed by recording the electroretinogram in response to both homogeneous flickering light (FERG) and contrast reversing gratings (PERG), respectively, 30 days after the indu a 60-minute episode of ischemia. Visual evoked potentials in response to contrast reversing gratings were also re to evaluate visual acuity and contrast thresholds. Cell survival after ischemia was assessed in retinal whole-mour stained with cresyl violet. Cats were intraocularly treated with NGF every other day, 3 times a week, for 30 days. were treated with either phosphate buffered saline or cytochrome c. RESULTS: After ischemia, the FERG was nc significantly affected. On the contrary, the PERG, visual acuity, and contrast thresholds were severely impaired. NGF treatment, PERG response amplitudes were much less reduced compared to controls, and visual acuity and contrast thresholds were virtually normal. In addition, a larger number of presumed RGCs was present in the NGI treated retinas compared to the cyt c-treated ones. CONCLUSIONS: The more proximally located retinal neurons particular RGCs, are highly vulnerable to ischemia. Intraocular NGF treatment was effective in enhancing the sur functional recovery of these neurons. This suggests that NGF may represent a novel therapeutic agent for the tre of ischemic ocular pathologies.

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